October 2006

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Cooperation has been a focus of intense interest in the biological and social sciences. Yet in spite of a tremendous effort to develop evolutionary models and laboratory experiments that explain the existence of cooperation in humans, relatively little effort has been invested in documenting the prevalence of large-scale cooperation in well-mixed populations and the extent to which it may be the result of biological or social forces. In this article we study voter behaviour as a form of cooperation that bears close resemblance to theoretical models in which individuals in a large population make anonymous decisions about whether or not to contribute to a public good. Matching public voter turnout records to an adult twin registry, we compare concordance in political behaviour between monozygotic and dizygotic twins. The results show that the decision to cooperate by choosing to vote is primarily determined by genetic factors. These results suggest that humans exhibit genetic variation in their tendency to cooperate and that biological evolution has played an important role in the development of political cooperation.

Voting is a classic example of a cooperation problem.[1] When one person votes, everyone with the same preferences benefits from the increased likelihood that their preferred outcome will result. Yet those who do vote must bear the cost of time and effort required to learn about the election and go to the polls. In large populations, the probability that a single vote will change the outcome of an election is miniscule,[2]

meaning that even very small costs to the individual typically outweigh the expected benefits to the individual. As a result, game theoretic models which assume individuals are self-interested and fully optimizing in their behaviour show that the equilibrium amount of cooperation (turnout) approaches 0 as the population becomes large.[3] Yet in spite of these predictions millions of people do vote, suggesting that something other than self-interest and optimizing behaviour drives their decision to cooperate. And the fact that millions of people abstain suggests that there may be inherent variation in the human tendency to cooperate.

Empirical models of turnout[4-6] typically contain numerous covariates including demographic factors (age, gender, race, marital status, education, income, occupational prestige, home ownership), attitudinal and behavioral factors (interest in the campaign, access to political information, strength of partisanship, feelings of civic duty, internal and external efficacy, political trust, church attendance, personal skill acquisition, humanitarianism, altruism, and patience), and institutional factors (closeness of the election, contact from political organizations, barriers to registration). Yet in spite of this everything-but-the-kitchen-sink approach, these models usually fit very poorly to the data. For example, one prominent model includes 32 variables but explains a mere 31% of the variance in turnout.[4] Moreover, the theories underlying these models completely ignore genetic or biological sources of variation. The implication is that voter turnout is purely an environmental phenomenon.

Recent work in neuroeconomics suggests we should look beyond the environment to explain political cooperation. In fMRI studies of behaviour in trust and social dilemma games, cooperation activates areas of the brain that have been linked with reward processing[7,8], suggesting that the brain has developed a mechanism to override self-interest in cooperation dilemmas. Cooperation in trust games also appears to increase in the presence of oxytocin[9] which reduces activation of the amygdala's fear

response.[10] Thus, variation in biophysical attributes like hormone levels and brain function may help to explain variation in cooperative behaviour. However, the neuroeconomics literature leaves open the question of whether biophysical differences in cooperation result primarily from environmental or genetic factors.

In order to estimate the degree to which cooperation is heritable, we study the turnout behaviour of (identical) monozygotic (MZ) and (non-identical) dizygotic (DZ) twins. MZ twins share 100% of their genes, while DZ twins share only 50% on average. Thus, if political cooperation is based in part on genetic characteristics, then MZ twins should exhibit more concordance (both twins vote or both twins abstain) than DZ twins. Moreover, if we assume that MZ twins and DZ twins share comparable environments, then we can use these concordances to estimate explicitly the relative importance of genetic, shared environmental, and unshared environmental factors (see Methods).

Some scholars have objected to the assumption in twin studies that MZ and DZ environments are comparable, arguing that MZ twins tend to be more strongly affiliated and more influenced by one another than DZ twins. However, studies of twins raised together have been validated by studies of twins reared apart,[11] and personality and cognitive differences between MZ and DZ twins persist even among twins whose zygosity has been miscategorized by their parents.[12] Moreover, contrary to the expectation that the environment would decrease concordance over time, MZ twins living apart tend to become more similar with age.[12]

To assess the impact of genetics on turnout, we obtained electronic voter registration records for 3.8 million voters from Los Angeles County with complete vote histories for 8 elections (three primary, two statewide, and three general) from 2000-2005 and matched them to the Southern California Twin Registry,[13] a volunteer adult

registry of MZ and DZ twins who live in the Los Angeles area. Records were matched by surname, first name, birthdate, place of birth, and zip code. Full matches were automatically included in our data. Partial matches on three or more of these attributes were manually checked and included in the data if the failure to match fully was determined to be the result of a typographical error. This procedure yielded vote histories and party of registration for 399 twins from same-sex pairs (172 MZ twins and 102 DZ twins in matched pairs, and 78 MZ and 47 DZ "singletons" where we found one twin in the pair but not the other—although singletons cannot help us estimate between-twin concordance, including these observations in the data does help us to estimate the mean turnout rate among twins for each election (see Methods).

A principal advantage of this approach is the use of field evidence based on third-party observations of actual voter behaviour rather than self-reports. This kind of data is rarely used in twin studies and is an especially important source for evaluating political cooperation since a significant number of individuals who did not vote typically report that they did.[14] However, there are also some limitations. About 30% of the adult population in Los Angeles County is not registered to vote, so we cannot include them in our sample. Since registration itself may be an act of political cooperation, this means our sample may be somewhat more cooperative than the population. However, focusing on registered individuals allows us to exclude those who might generate false concordance because they are ineligible to vote due to foreign citizenship status or criminal records. We also note that mean turnout rates were slightly higher in our sample than in the population, though turnout rates between MZ and DZ twins did not differ significantly (see supplementary information).

Results

Mean concordance in the pooled turnout observations using polychoric correlation was significantly higher (p=0.006) in the MZ twins (0.71) than the DZ twins (0.50).

However, naïve correlation measures are only a crude guide since they make no provision for multiple observations of each twin. Instead, we conduct a mixed-effects Bayesian ACE analysis which uses observed turnout decisions to estimate a latent tendency to cooperate for each twin, which itself is a function of latent heritability (A), common environment (C), and unshared environmental (E) factors. We then rescale the estimated ACE factors to sum to one in order to estimate what portion of variance in the tendency to vote is due to each factor (see Methods).

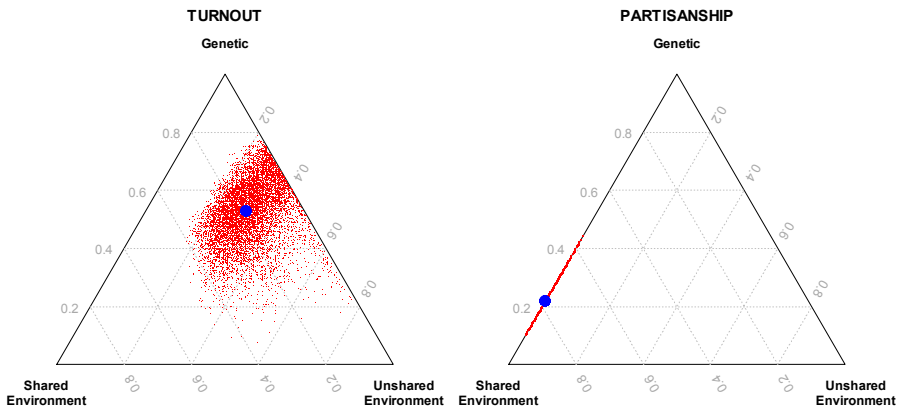

Figure 1. Ternary diagram of 10,000 draws from posterior Bayesian distribution of estimated components of variance in voter turnout (left panel) and party affiliation (right panel). The probability that the true coefficients lie outside the region of these draws is p=0.0001. The mean of the distribution is noted by the blue solid circle. These results suggest that heritability plays a significant role in political behaviour. Variation in political cooperation (turnout) is more than half heritable, while variation in political affiliation (partisanship) is only about one-fifth heritable.

Fig.1 shows the posterior distribution of the estimated factors. About 53% (95% C.I. 23%,73%) of the variance in turnout behaviour can be explained by genetic factors. The environment also plays a role, but the unshared environment accounts for much more variance (30%, C.I. 22%,39%) than the shared environment (17%, C.I. 1%, 43%). These results are based on data that pool same-sex male and female pairs. When we conduct the same analysis on each gender separately we do not find significant differences in the estimates. The genetic component for males is 51% (95% C.I. 6%,79%) and for females is 59% (95% C.I. 19%,81%).

To check the validity of the method we used to collect the data and conduct the analysis, we also estimated a mixed-factor Bayesian model of affiliation with a political party. Fig.1 shows that genetics plays only a small role in partisanship, explaining just 22% of the variance (95% C.I. 14%,36%). This result is consistent with earlier findings in the Virginia 30k study that show genes explain about 14% of the variance in self-reported party affiliation.[15]

Note that the contrasting findings for voter turnout and party affiliation would be difficult to explain with the hypothesis that parental or mutual influence causes MZ twins to be more alike than DZ twins (i.e. their shared environments are not comparable). If so, then we would need to be able to explain why this influence is so strong for political cooperation and so weak for party affiliation, which would be especially puzzling in light of vast evidence that party affiliation is primarily learned from one's parents.[16] We also note that in our sample MZ twins are actually *less* likely than DZ twins to live at the same address (40% vs. 49%) or in the same zip code (50% vs. 52%). Thus, greater concordance in MZ twins is probably not due to higher frequency of contact.

Discussion

To a certain degree, these results should not be surprising. Scholars have already documented a genetic basis for altruism and prosocial behavior[17,18] and these behaviours in turn have been linked to voter turnout.[19-21] However, social scientists (outside of the field of psychology) have been extremely reluctant to admit a role for genetic and biological factors in political phenomena which has biased scholarly interpretations of several important phenomena.

For example, if political cooperation is determined in part by genetics, it would conform to two well-known features of voting behaviour. First, parental turnout behaviour has been shown to be one of the strongest predictors of turnout behaviour in young adults.[4] Although this has previously been interpreted as the result of social influence, the findings here suggest it may be mostly due to heritability since the shared environment appears to play only a small role. Second, turnout behavior has been shown to be *habitual*—people typically either always vote or always abstain.[4,22-26] Scholars previously interpreted this as the result of reinforcement learning, but given the small effect of environmental variation it might also be primarily due to inherent genetic variability.

More importantly, our results have general implications for the vast literature on cooperation in humans. Although previous laboratory studies examined cooperation *between* twins, [27,28] none has focussed on the question of how twins cooperate with unrelated members of the general population. Thus, this article represents the first attempt to estimate the genetic basis of cooperative behaviour in large, well-mixed populations. The results suggest that humans are endowed with genetic variation in their tendency to cooperate, which supports theoretical models in which cooperators and noncooperators coexist. This would tend to favour evolutionary models which yield polymorphic equilibria[29] or heteroclinic cycles[30] over those that predict monomorphic populations of cooperators or defectors.

Our results also support models which suggest the evolution of cooperative behaviour is not merely the result of social evolution—instead, cooperation may result from the co-evolution of genetic and cultural characteristics.[31] Although here we assume genetic and environmental factors are additively separable, the strong influence of genetic factors suggests that more detailed models of the interaction of genes and culture (G x E models) are likely to identify a significant role for genes in the evolution of cooperation.

Finally, our results have important implications for recent work in neuroeconomics. Scholars have linked several areas of the brain[7,8] and some hormonal responses[9,10] to the tendency to cooperate. However, these studies do not indicate whether variation in cooperation across individuals is due to environmental or genetic influences on the physiological development of the brain and endocrine systems. Our results suggest an important role for genetic evolution in the development of physiological systems that contribute to an individual's willingness to cooperate.

Methods

The most popular method of fitting variance component models to twin data is maximum likelihood estimation of structural equations models. An alternative approach is to formulate the twin model as a linear mixed effects model if the phenotype is continuous or a generalized mixed effects for a discrete phenotype.[32,33] Generalized mixed effects models are simply generalized linear models with continuous random effects. Models of discrete phenotypes present computational challenges for structural equation modelling software packages because the likelihoods contain integrals that cannot be evaluated in closed form and thus must be evaluated numerically. The maximum likelihood estimation of complex models with many random effects requires repeated numerical evaluation of high-dimension integrals.[33]

As a result, researchers have begun to use Bayesian models implemented using Markov Chain Monte Carlo algorithms. These algorithms evaluate the high-dimension integrals using random draws rather than directly.

The application of Bayesian inference to genetic models has begun to receive widespread attention within the literature. Recent studies have successfully applied Bayesian methods to binary data,[33] survival analysis,[34] nonlinear developmental change and GxE interaction,[35] item response theory,[36] longitudinal models,[37] and multivariate models for ordinal data.[38]

Simple polychoric DZ and MZ correlations for the sample suggest the appropriate model for turnout should account for additive genetic, shared environmental, and non-shared environmental effects. This model is known as an ACE model. We can specify this model as a generalized linear fixed-effects model where subject j is a member of family i and voting in election k. The link function g reflects the fact that we believe the underlying variable, the propensity to vote, is a continuous variable that we are only able to measure categorically.

$$g(\text{MZ Phenotype}_{ij}) = \mu_k + A_i + C_i + E_{ij}$$

$$g(\text{DZ Phenotype}_{ij}) = \mu_k + A1_i + A2_{ij} + C_i + E_{ij}$$

The μ_k is a parameter that controls for election-specific variation in voter turnout. A1 and A2 are additive genetic effects, C is the shared environmental effect, and E the unshared environmental effect. In mixed models A1, A2, C, and E are modelled as random effects and μ as a fixed effect. The additive genetic effect is split into two constituent parts to account for the difference in genetic covariance between MZ and DZ twins (the genetic variance is the same for MZ and DZ twins, but the genetic covariance for MZs is twice as large as DZs because they share twice as many genes). As in previous literature, we assume that the random effects are normally distributed:

A1~N(0, var(A)/2), A2~N(0,var(A)/2), C~N(0,var(C)), and E~N(0,var(E)) where A is the total additive genetic effect. We use vague priors to ensure they do not drive model results (a mean-zero normal distribution with variance 1,000,000 for μ and a uniform distribution on [0,1000] for the square root of var(A), var(C), and var(E)).

We employed a threshold model assuming a normally-distributed latent propensity to vote.[33] This latent variable is linked to the observed decision to vote or abstain via a step function. Because the dependent variable is dichotomous, the total variance of the latent variable must be fixed for identification. We achieve this by constraining the variance of the unshared environment var(E) to equal one and then using the estimates of var(A) and var(C) to derive the proportion of variance generated by each factor. We also estimate the population means by fixing the threshold to zero. To detect convergence in the estimates we ran two MCMC chains and applied a Brooks-Gelman test to the ouput.[39] The 'potential scale reduction factors' for each variable in our simulations were reduced to 1.1 or less by the 40,000th draw, indicating convergence. To sample the posterior we discarded the first 50,000 draws and kept every 100th draw from the next 500,000 draws from each of the two chains. Test results drawn from 2 million draws yielded identical estimates.

To analyze party affiliation, we follow the same procedure as for turnout, except there is only one observation per individual so we set k={1}. A substantial number of individuals reported no party affiliation and in rare cases they reported party affiliations other than the two major parties. The results reported in the paper treat party of affiliation as a dichotomous variable to maintain comparability with results from the Virginia 30k (Republicans are coded as 1 and others as 0, but identical results are obtained when we code Democrats as 1 and others as 0).

Supplementary material

	3/2000 primary	11/2000 general	3/2002 primary	11/2002 general	10/2003 statewide	3/2004 primary	11/2004 general	11/2005 statewide
All twins (*N*=399)	0.54	0.76	0.36	0.57	0.68	0.44	0.84	0.46
MZ twins (*N*=250)	0.55	0.75	0.36	0.58	0.71	0.42	0.86	0.49
DZ twins (*N*=149)	0.53	0.79	0.37	0.56	0.62	0.46	0.80	0.42
Population (*N*=3.8 million)	0.48	0.68	0.26	0.45	0.55	0.38	0.79	0.47

Table 1. Comparison of turnout rates in twin sample and general population in Los Angeles County, by election

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